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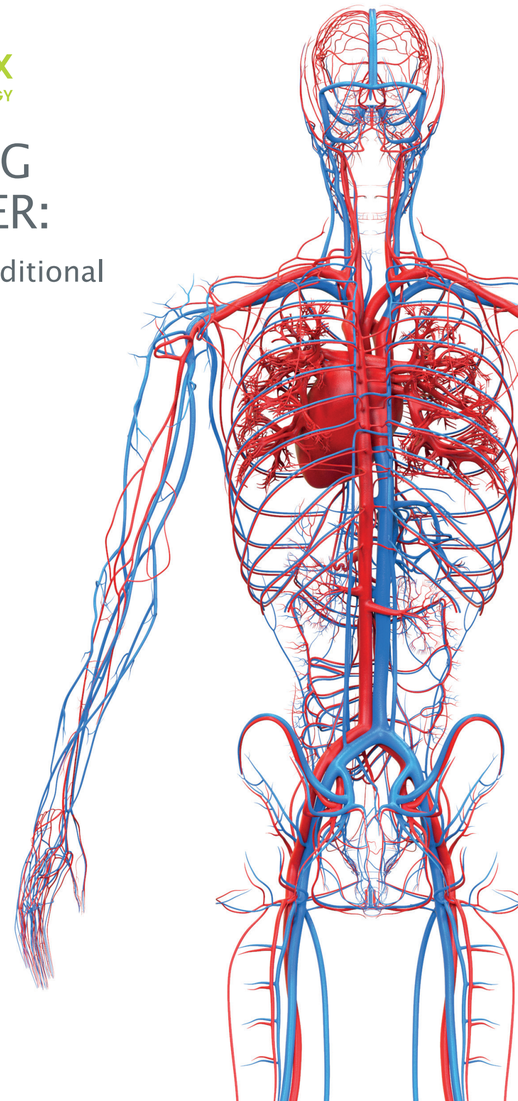
DEVELOPING IPC FURTHER:

Why IIC offers additional advantages



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DEVELOPING IPC FURTHER: WHY IIC OFFERS ADDITIONAL ADVANTAGES

One of the most effective and gentle ways to improve micro- and macrocirculation – this is true for devices for intermittent impulse compression (IIC) like VADOplex*, that are an advanced version of intermittent pneumatic compression (IPC) devices. IIC extends the IPC's possibilities by utilizing basic principles of human physiology.[1] These correlations and thus the IIC's special mode of operation were demonstrated by the British surgeon A. M. N. Gardner and his compatriot, the radiologist R. H. Fox, and described in their angiology reference book „The Venous System in Health and Disease". Both are considered inventors of IIC and have advanced its clinical application significantly. Their most important findings are summarized in this overview.[2]

THE 0.4-SECOND IMPULSE AS A DECISIVE ADVANTAGE

IIC devices – unlike the related IPC devices with their slow pressure build-up – generate a very fast pressure impulse that focuses on the sole of the foot's or the hand's venous plexus.[1,2] With VADOplex, this pressure build-up takes less than 0.4 seconds.[1] Thereby, IIC simulates the physiological effect of natural movement. That is, it empties the venous plexus and deep veins of the lower extremities. Thus, IIC accelerates venous return and increases arterial inflow. This IIC-associated acceleration of blood flow can be demonstrated using Doppler frequency spectrum analysis.[2]
Here comes the crucial additional effect that is the advantage over the IPC devices with their slower impulse build-up: with its suddenly accelerated pressure impulse, the IIC creates a turbulent flow in the veins.[2] This generates shear forces that act on the venous endothelium and support the body's own production of 2 important vasoactive tissue hormones:[2]

- ✓ **Nitric oxide (NO), or endothelium-derived relaxing factor (EDRF)[2]**
- ✓ **Prostacyclin, or prostaglandin I_2 (PGI_2), a derivative of arachidonic acid[2,3]**

Their release – especially of NO – makes IIC special as NO and prostacyclin enhance the effect of pneumatic compression:[2]

- ✓ **NO and prostacyclin improve thrombosis prophylaxis.[2]**
- ✓ **NO and prostacyclin increase microcirculation.[2]**
- ✓ **NO improves pain relief.[2]**

NO AND PROSTACYCLIN: 2 ENDOGENOUS SUBSTANCES AT A GLANCE

NO and prostacyclin act synergistically:
- Antithrombotic by inhibiting platelet aggregation.[2] NO also inhibits monocyte adhesion[4,5] and increases fibrinolysis.[6]
- As vasodilators and thus regulators of blood flow. NO has a more potent dilatory effect than prostacyclin.[2]

Additionally, NO[2]
- has an analgesic effect and
- promotes bone healing after fractures.

Based on these differences, both substances complement each other in their effect:[2]

	Nitric oxide (NO)	Prostacyclin
Molecular size	Very small - spread by diffusion in the tissue (convective transport)	Large - less diffusion into the tissue
Physiological half-life	6 to 9 seconds – (oxygen-dependent). (prolonged under hypoxia and shortened to 0.1 seconds under high oxygen).	Up to 4 minutes

SPECIAL FEATURE OF IIC: ADDITIONAL EFFECT DUE TO NO AND PROSTACYCLIN

IIC acts on the venous and arterial system as well as on the tissue. Thereby, the tissue hormones NO and prostacyclin – generated under the influence of the impulse application – intensify the physical effect, whereas IPC devices with their slower impulse build-up act almost exclusively mechanically.[2]

1. In the veins: Enhanced thrombosis prophylaxis
The IIC's antithrombotic effect is based on 4 mechanisms:

- IIC simulates the effect of physical exercise, accelerates venous return, creates turbulence in the pockets of venous valves, and thus prevents stasis.[2]
- The tissue hormones NO and prostacyclin, which are produced only during IIC, inhibit platelet aggregation and thus initiation of thrombus.[2,7-9]
- NO prevents monocyte adhesion.[4,5]
- NO increases fibrinolysis.[6]

2. In the arteries: Increased microcirculation
The IIC's pressure impulse reduces temporarily (20 seconds) vascular congestion by about 30% and increases arterial blood flow (hyperemia). Thereby, several mechanisms interact:[2]

- Dilatory NO plays a decisive role. It is released in IIC – but not in IPC with its slower impulse. NO is produced in the veins under the influence of the pressure impulse, diffuses through the tissue to the neighboring arterioles, relaxes their vascular musculature, dilates the arterial vessels and thus leads to hyperemia. Subsequently, NO is depleted – regulated by oxygen concentration.[2]
- With repeated impulse compression, arterial blood flow increases progressively over 5 minutes or longer.[2] Moreover, the additionally produced prostacyclin (longer half-life) has a synergistic effect.[2,10]
- It might also be possible that the pressure impulse releases aggregated leukocytes, which likewise impede the microcirculation, and thus has a further positive effect.[2]

The crucial contribution of NO and prostacyclin is supported by 2 observations:[2]

- IIC-associated arterial blood flow in the extremities is exclusively diastolic and occurs even during arterial blockade. The extent of hyperemia depends on venous "priming".
- Administration of prostacyclin inhibitors shortens the duration of hyperemia after impulse compression.

NO also reduces the oxidation of low-density lipoprotein cholesterol (LDL-C) and thus counteracts atherosclerosis.[11] Increased microcirculation and oxygenation (nutrition) of the tissue in turn improve wound and bone healing.[12-14]
In addition, NO is involved in bone formation (callus formation) after fractures.[2]

3. In tissue: improved pain relief
On the one hand, the IIC-induced hyperemia relieves ischemia-related pain. On the other hand, IIC reduces trauma-associated pain – via several suspected mechanisms:[2]

- Reduction of venous congestion leads to a decrease in often painful swelling, counteracting acidosis that increases pain sensitivity.
- The IIC-generated NO also has analgesic effects.

Thus, data from studies show that IIC relieves trauma-related pain better than cryotherapy.

How IIC affects the lymphatic system[15]
Following the basic work of Gardner and Fox, knowledge of IIC has expanded with regard to the lymphatic system and swelling reduction. Again, several mechanisms interact:

- Emptying of the venous plexus and accelerated venous return decreases blood capillary pressure. This results in venous resorption of the edema fluid that is potentially present in the tissue.
- The profound lymphatic vessels, which have no motor function of their own, also benefit from the pressure impulse. IIC propels the lymph and thus contributes to decongestion.
- In addition, it is believed that the NO released during IIC reduces the contraction of the lymphatic pump, causing the lymphangions to fill more, which can thus remove a higher volume of lymph. As a result, a suction is created in the initial lymphatic vessels, which eventually promotes lymph formation.

AT A GLANCE: THE ADDITIONAL EFFECT OF IIC[1,2]

IIC (VADOplex®)	IPC with slower impulse build-up
technological principle	
Pressure impulse focuses on the sole of the foot's or the hand's venous plexus.	Broad compression pressure on the tissue.
Very fast build-up of the pressure impulse within less than 0.4 seconds.	Slow pressure build-up.
physiological effects	
Suddenly accelerated blood flow generates shear forces acting on the inner surfaces of the veins (venous endothelium).	No sudden acceleration of blood flow.
Shear forces promote the production and release of tissue hormones by the venous endothelium - especially endogenous nitric oxide (NO) and prostacyclin.	Significantly lower production and release of tissue hormones.
medical mechanisms of action	
Physical and pharmacological mechanism of action: NO and prostacyclin extend and enhance the effect of pneumatic compression in terms of thrombosis prophylaxis, microcirculation, pain relief and swelling reduction.	Only physical mechanism of action through pneumatic compression.